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PHYSIOLOGICAL PLANT PATHOLOGY[1]

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For the purpose of the present discussion physiological pathology is not distinguished from pathological physiology. Plant pathology is considered merely with respect to certain scientific aspects of the subject, and as a field of work dealing primarily with a few classes of diseases in vascular plants.

It is almost universally conceded that disease is exhibited through abnormal cellular activity. This concession establishes at once all manner of pathological phenomena in a definite theoretical relation to physiology. Normal and abnormal physiology are relative terms and they may be said to emphasize different types of biological equilibria; but the distinctively abnormal condition may be adequately studied only through comparison with that condition essentially normal, so that from many standpoints these terms are largely formal distinctions, and in representing lines of scientific inquiry, at least, they converge and become a common direction of investigation.

It is no new idea, but it is worthy of repeated emphasis, that disease is a complex response, or type of behavior, of cells and groups of cells. The response is manifestly produced by a stimulus of some type, and it is either external or internal. It is therefore clear that whatever other relations may be considered important in the outlying applications of plant pathology, the basis of the whole science rests upon the foundation-stone of cellular irritability. It seems also evident that competency in the study of the real nature of disease means physiological competency.

Is physiology made the basis of any considerable part of present scientific plant pathology? If attention be directed merely to those diseases which are not induced by specific organisms, the physiological relation is more or less prominent. It be required, however, that we examine the present-day extent of physiological study not merely in

[1] Read before the Botanical Society of America, Minneapolis, Dec. 29, 1910. This was the second of three papers in a Symposium on Some Aspects of Plant Pathology.

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the so-called physiological diseases, but especially in the better known aspects or current relations of plant pathology. All who have followed the developments in this general field must admit that the investigations are and have been primarily concerned with diseases induced by parasitic bacteria and fungi. The fungi and bacteria are therefore a most important part of the disease relations; but these organisms themselves and the economic extent of the ravages are too often regarded as practically the only essential features of pathology. The study of these organisms has been most timely; and the fact that they have as agents received almost exclusive attention is the first logical outcome of their importance.

Referring to the diseases of higher plants brought about by parasites, I have purposely limited the field here touched upon to fungi and bacteria. In this connection it seems well to mention the unfortunate circumstance that custom generally continues to eliminate from the category of diseases inviting consideration by the plant pathologist those varied and often remarkable changes in tissues induced by insects and other minute animals. The attitude of pathologists, nevertheless, is generally to ignore this interesting field, where the growth correlations are among the most striking known.

DeBary, who may be regarded as the founder of modern plant pathology, both through his epoch-making advances in the study of life histories of fungi, and of fungous parasitism, gave direction to a wave of investigation, which, sweeping forward under the influence of many new forces, presents to-day its maximum power. Although his influence was important in developing pathological methods, DeBary emphasized morphology, and even this emphasis is yet dominant. There has been brought to the type of work which he instituted the assistance of all those devices which might throw light upon morphology and life history, and rather more incidentally upon some of the physiological relations. Such studies as have been made in the latter direction are more commonly with respect to the physiology of the parasite. In order to see that this is essentially true we have only to review in the literature the work which has been done with respect to a few classes of the fungi. First, among those diseases induced by Phycomycetes, this may be seen to advantage. The incomparable studies of DeBary upon *Cystopus*, and other forms, and those of Farlow a few years later upon *Plasmopara viticola* are models of mycological research, and pathological research has gone but little farther. Far more is known of the rôle of the Ascomycetes and imperfect fungi as disease producers to-day; but only in exceptional cases have we been afforded vistas of new problems in which the host is involved. Some studies upon the

mildews, in particular, are among the exceptions which will suggest themselves.

Recent studies upon the rusts develop many mycological problems ; but those results of most physiological significance are such as the studies of Marshall Ward and others, giving facts respecting the relative immunity of certain hosts, or the specialization of forms of the parasite. The mycoplasm hypothesis has directed some study to the cell relations of the rust fungi ; but the actual disease conditions are indeed little known. The obligate parasitism of the rusts, the stimulation of the host, frequently in the production of hypertrophies and witches brooms, are suggestive of the problems.

In the studies upon the Basidiomycetes there may be found a mass of mycological facts ; but more interesting in this connection are those relatively few data serving to indicate the great capacity of these organisms, once within the tissue, to decompose or digest the varied types of organic matter of which plant bodies are constituted. In this group of organisms, which, for the most part, seems to have little direct association with the living cells of the hosts, the physiological problems are perhaps the simplest.

The remarkable development of bacteriology has promised much in the way of introducing the physiological method in plant pathology, since physiological characters are essential in a proper description of the organism, and in the establishment of its pathogenicity. With bacteriological methods there has come, in addition to an accurate procedure for the determination of the causal organism, a more thorough study of the disease lesions ; and it is to the great honor of plant pathology in this country that the bacteria as agents of plant diseases have been so admirably studied as through the work of Erwin F. Smith and others. The bacteriological method in general has greatly advanced the status of pathology. The necessity of studying the peptonizing, hydrolyzing, or other fermentative capacities of the bacteria is stimulating a general desire to go further, although temporarily many may apply chemical and microchemical study to the parasite alone. For a complete study of diseases and the fundamental causes of diseases the physiological study should not halt at the threshhold of the host relations.

It is not intended in any way to underrate the advances already made in pathology. Every growing season makes known numerous new disease-producing organisms ; and many of the obscure riddles of life history have been skillfully guessed in recent times. There have been developed, even within the past few years, decided improvements in the methods of controlling disease organisms, and this especially

has brought to the work a stupendous popular interest and a certain financial support. The advances, nevertheless, are along the old lines, and the old lines must remain prominent in the general subject. It is not sufficient, however, that plant pathology should represent an advanced type of mycological study, likewise a certain perfection of diagnostic and etiologic technique, together with an effective system of prevention or control. Every disease produced by an organism presents the definite problem of certain complex relations between the cells of the host and those of the parasite. This is the essential feature of the physiological plant pathology of the fungi and bacteria. There is ample opportunity yet for the mycologist along the old lines, and more than ever for the practitioner along new lines; but this distinctive field of physiological pathology is also open for more consecutive, painstaking, and systematic effort. The question as to whether the physiologist or pathologist shall undertake such studies, training therefor being granted, is a secondary matter; it is the nature of this field of work which is of first importance.

The problems which will confront the investigator are undoubtedly distinctive and difficult to approach. Often the methods must be strictly physiological and chemical. Some hints respecting the probable directions of the work may be gained from a knowledge of the relatively extensive accomplishments in the physiological pathology of human and animal diseases. In this latter field, physiological pathology and chemical pathology are no longer merely theoretical directions. In spite of all the advances made in animal pathology Verworn sees in this subject too feeble a tendency toward physiological study. What, then, may be said as to the comparative vigor of the equivalent plant work?

There is insufficient knowledge respecting the nature of the responses in the cells of the host when invaded by the parasite. One common type of response, induced both by parasitic organisms and perhaps by other causes as well, is manifest in the proliferation of merismatic cells, resulting in the production of abnormal swelling or growths. Among the abnormalities caused by fungi and bacteria are those characterising club-foot of cabbage, plum-pockets, crown-gall, and numerous other familiar diseases. While such hypertrophies are entirely abnormal, the cells constituting these may or may not undergo unusual modification. The swelling is not necessarily accompanied by any direct degeneration phenomena. Any facts throwing light upon the nature of the stimuli producing such structures would be most important, for the greatest ignorance prevails respecting the causes of unusual cell division generally. The factors would seem to

be entirely different from those conditioning oedema; but this is not certain. M. H. Fischer has recently brought to the assistance of certain studies upon oedema in animals many suggestive experimental results from the field of colloidal chemistry. These results, in brief, emphasize the important water-relations of colloids and the conditions affecting these relations.

In his recent work upon gummosis of Prunus and Citrus, Butler has pointed out an interesting interrelation of factors. He says, "For gummosis to develop two conditions must be simultaneously fulfilled. The cambium must be actively laying down new tissue elements, and a superabundance of water must be present in the soil." It is therefore rapid growth and an abundant water supply which are ultimately the important internal factors rendering the tissues susceptible to gummous degeneration. Gummosis may occur autogenously, but practically it does not. It requires a releasing stimulus, or, perhaps more properly, an agent which may vastly increase the velocity of the reaction. The stimulus generally consists in some form of injury, and this may be effected by fungi, insects, deleterious chemical agents, or burning.

The effect of *Cystopus candidus* upon the tissues of its host is commonly different from that of such related parasites as *Phytophthora infestans* or *Pythium DeBaryanum*. The first mentioned usually stimulates to abnormal cell multiplication, while the others kill, or kill and disorganize. It is rather remarkable that the entire group of rusts show very little tendency to kill immediately the tissues which they invade. There is no general hydrolytic or breaking down process, and there is every reason to believe that whatever the nature of the stimulus which these fungi produce it is not, at least, of enzymic nature. Degeneration phenomena are, of course, characteristic of the action of many fungi. It appears to be clearly demonstrated by many investigators that certain fungi invade the cells of the host after the latter are killed by the penetration of acid substances excreted by the fungus. The digestion of certain cells and ultimately of tissues may then follow. In this case there is apparently no contact of the parasite with living protoplasm. There are, however, all grades of parasitism, and there must be a variety of effects induced in the host, including changes essentially autolytic. In general, the physiology of penetration is poorly understood, and it will reward investigation.

Upon entering the host, certain fungi and bacteria may accomplish a phase of their work through the secretion of the enzyme pectinase, causing the digestion of the middle lamellae of the host cells. The result of such action has long been known, and recently L. R. Jones

has studied the phenomenon in some detail with respect to the important pathogenic form *Bacillus carotovorus*. Solution of the middle lamellae is accompanied by other disturbances. The capacity to produce this enzyme, however, is not alone sufficient to determine or maintain the parasitic habit, for certain saprophytic bacteria and fungi are similarly capacitated. There must be other products causing an intoxication or death of the cells and ultimately a digestion of much besides middle lamellae. It is necessary to develop further facts respecting the factors in these complex relations.

It is now well demonstrated in animal work that proteins, enzymes, and other organic bodies (toxins) of one organism introduced into the tissues of another may be distinctly injurious. It is now precise information which is needed with respect to plants. Some use may be made of the animal work, but the conditions are different, and it is only possible to gain general ideas for certain of the relations and principles involved in plant study.

From the beginning of pathological study the effects of various environmental (generally climatic) factors, such as humidity and temperature, upon the germination of spores and spread of disease has been so obvious that we may have considered these almost exclusively. It is certain that climatic factors are important in conditioning the germination of fungous spores, and usually, when these spores are superficially produced, in facilitating spore production as well. On the other hand, climatic factors are always important in determining the composition of plants. We must suspect that composition of the cell sap is important directly or indirectly in conditioning effective penetration, so that cognizance must be taken of the changeable nature of this cell sap. Fortunately, there are already suggestive indications. Astruc has shown that in non-succulent vegetation generally relative acidity is greatest in the organs of more vigorous growth. Therefore, maturing organs show a minimum of oxidation and acidity. On the other hand, there is considerable variation with changes in external factors. In succulent plants such as the Crassulaceae the state of growth of the organ is not so important, but the slightest changes in the environment, through changed metabolism, may induce considerable disturbances in the relative acidity.

Injuries and stimulation of various types are doubtless equally important in the production of such differences in composition as those above referred to. Not only is relative acidity important, but other metabolic differences as well.

The experimental results thus far reported seem to indicate that partial shade reduces relative abundance of volatile oils, sugar content,

and protein accumulation in plant tisssues, as compared with bright light. It is therefore especially interesting that observations tend to show that predisposition to many diseases is a factor of changes in one or more conditions of the environment. Soil factors as well as climatic factors are held at least indirectly responsible. It is, however, unnecessary to review here specific instances. This type of predisposition may be looked upon as induced predisposition. It may be typified by the effects of injury, as in the well-known cases where certain naturally resistant varieties or species of grasses are rendered subject to mildew by wounding the leaf at a distance from the point of attack. A variety of environmental factors act indirectly upon metabolism, and in attempting to relate the indirect effects of conditions upon predisposition it will be found that physiological or chemical investigation in this direction is so limited as to afford an insufficient basis for any general hypothesis. Blackman has drawn attention to the fact that "the way of those who set out to evaluate exactly the effects of changes in a single factor upon a muli-conditioned metabolic process is hard."

It has also been shown that certain fungi are very sensitive to conditions. High summer temperature is supposed to limit the destructiveness of *Phytophthora infestans*. In this connection the work of Balls is suggestive, and may be cited. He observed that the cotton 'sore shin' fungus grown on agar at high temperatures (34° C) produced an abnormal mycelium. On hard agar there was rapid inhibition of growth; on soft agar inhibition with increase in the time factor; and in liquid media the effect was much delayed, the delay being greater as the volume of the liquid was increased. His explanation is 'that some substances (or mixture of substances) is formed by the protoplasm in katabolism, and inhibits growth by its accumulation.' The hypothetical katabolite, designated X, is diffusible; and media containing it inhibit growth. He states that 'the decrease and ultimate cessation of growth at high temperatures, is due to the accumulation of katabolic products in the cells.'

Predisposition or resistance to disease may be hereditary. Since this relation is to receive special consideration in the program, it is here sufficient to note that there is now at hand ample material for a thoroughgoing study of some of the factors governing resistance and susceptibility. The problem is doubtless extremely difficult; but it is believed by some that the work may yield results. Available material includes among notably resistant plants *Triticum monococcum*, which is resistant to rust; certain American grapes, resistant to the mildew; and strains of the common cowpea and of flax, which are only slightly attacked by certain disease-producing species of Fusarium.

Recent study indicates the development of a form of red clover fairly immune against Colletotrichum, and the Pollock strain of Rockyford melon promises considerable immunity to the Macrosporium disease. It may be stated as a general fact that where there are many varieties of the host there will be exhibited grades of resistance. There are naturally immune varieties, and likewise those produced by means of hybridization and selection. At present there is no information respecting the cause of this difference in behavior as regards resistance.

The advancement of physiological pathology is dependent upon the work of the physiologist, of the pathologist, and of the biochemist, or upon adequate consideration of the several view-points which these names represent. The problems are fundamentally difficnlt, and many of these invite united effort.

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A PRELIMINARY REPORT ON THE EFFECTS OF ARSENICAL COMPOUNDS UPON APPLE TREES[1]

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(WITH PLATES XVI AND XVII.)

Since Dr. Headden[2] in 1908 called attention to the serious injury to the crowns of fruit trees due, in his judgment, to arsenical poisoning, both entomologists and plant pathologists have shown a keen interest in the question of whether or not we are jeopardizing the lives of our trees by applying each year heavy sprays of arsenical insecticides. It is indeed a vital question, for if the orchardists cease to use this class of insecticides, with no equivalent substitute, there will be a heavy loss from wormy fruit; while on the other hand, if they continue to use these spraying compounds and thereby injure the orchards throughout the country, or even the West, to the extent reported for Colorado, the aggregate loss through the killing of trees will be appalling. Not all are agreed, however, as to the soundness of Headden's conclusions. Without going fully into the history of the controversy, we may note that Ball[3] rejects the idea of arsenical poisoning, referring the death of the trees to seepage and alkali, while Grossenbacher[4] would have us believe that the real cause is some form of winter injury. Headden[5] in a latter bulletin, however, maintains just as stoutly as before and with additional evidence that arsenical compounds are corroding the crowns and causing general injury to the trees, and Gillette[6] suggests the possibility of substituting arsenic trisulphide for the arsenicals now used as insecticides, it being less soluble in water.

In the spring of 1909 the writers of this paper inaugurated at the Montana Experiment Station an extensive series of experiments on the general problem of the effects of arsenical compounds on plant life. Practically every known compound of arsenic is being tested on a great

[1] Read at the Minneapolis meeting Dec. 29, 1911.

[2] Headden, Wm. P. Arsenical Poisoning of Fruit Trees. Colo. Exp. Sta. Bull. 131: 1908.

[3] Ball, E. D. Is Arsenical Spraying Killing Our Fruit Trees? Jour. Econ. Ent. 2: 142. 1909.

[4] Grossenbacher, J. G. Crown Rot, Arsenical Poisoning and Winter Injury. N. Y. (Geneva) Exp. Sta. Technical Bull. 12: 1909.

[5] Headden, Wm. P. Arsenical Poisoning of Fruit Trees. Colo. Exp. Sta. Bull. 157: 1910.

[6] Gillette, C. P. New Sprays for the Codling Moth. Jour. Econ. Ent. 3: 29. 1910.

variety of plants, both higher and lower, under a wide range of conditions. The work is expected to extend through a considerable number of years and this preliminary report takes up only a limited phase.

The experiments here recorded were carried on in the college orchard where spraying for the codling moth has not yet become necessary, and where no arsenical compounds had ever been applied. For this reason it was deemed wise to prevent the contamination of the ground with these compounds by using the precautions described below. This work was begun in June, 1910, and is being continued. The problems about which the experiments centre are: First, can the arsenical compounds commonly used as insecticides injure the bark of fruit trees? Second, which of these compounds are most and which least injurious? Third, what is the nature and extent of the injury? Fourth, what conditions most favor the injurious action?

The following compounds were applied:

Arsenic acid, pure, Merck.

Arsenic trioxide, highest purity, Merck.

Arsenic disulphide, Merck.

Arsenic trisulphide, technical, Merck.

Calcium arsenite, Merck.

Lead arsenate, technical, Merck. (a mixture of acid- and ortho-arsenates).

Paris green, Ansbacher & Co.

Sodium arsenite, pure, Merck.

Zinc arsenite, (ortho.) California Spray Co.

The tests are to be repeated and extended next season with specially purified and analyzed chemicals prepared to order by the J. T. Baker Chemical Co. The arsenic acid and sodium arsenite are, of course, not used as insecticides because of their ready solubility but were included in these tests as checks on the others and also because they may sometimes be found as impurities in the other compounds.

These chemicals were applied to branches varying from ¼ to 2½ inches in diameter, and to crowns from 3 to 5 inches. In the treatment of the branches the so-called "insoluble" compounds, in weighed amounts, were spread on small squares of absorbent cotton which were wrapped about the limbs in such a way that the chemicals made a fairly even layer all round for a distance of about 4 inches. These cotton bandages were then bound firmly in place with surgeon's gauze and wet with distilled water. In order to prevent any dripping to the ground of the arsenical suspension, metal boxes were attached to the limbs close under the bandages. To make these boxes more effective, limbs were selected that showed a decided sag at one point and the

chemicals were applied at the lowest portion, so that the liquid could not follow along the limb away from the bandage in either direction without runnning up hill. It must therefore necessarily drip down into the metal box. Owing to the slight and irregular rainfall in this locality, the bandages were wet with distilled water every day at about five p. m. This served to keep them moist practically all of the time. Not enough water was added, however, to cause more than a very few drops at most to run out. It was intended by means of the bandages to roughly simulate the physical conditions, especially as to moisture, found about the crowns of the trees.

The soluble compounds were applied in a similar way, excepting that the bandages were put on first and the chemicals then added in solution. These were also kept moist with distilled water. In a few instances the evaporation and possible drip were prevented by wrapping the bandages tightly with dental rubber dam, in which cases the boxes were omitted. This proved highly satisfactory except that the rubber became friable in a few weeks unless protected from the sun by an outer covering. It is needless to say that in an orchard where the soil already contains much arsenic from spraying, these precautions against dripping would be unnecessary, excepting to protect the foliage below the bandages, unless soluble compounds or very heavy applications of insolubles were used.

The crowns were treated by digging a shallow trench close around the tree, partly filling it with the chemical suspended in water, and replacing the earth. No attempt was made in these few cases to keep the arsenicals from mingling with the soil. The trees selected were, however, mostly in the lowest part of the orchard.

A total of 55 limbs and 10 crowns have been treated as indicated above and the results form the basis for this paper.

In the earliest experiments upon the branches some twigs were removed where the bandages were to be applied and it was also observed that others had previously been cut off in pruning. Careful note was made of these wounds and the results subsequently observed prove their significance even greater than was anticipated.

A brief description of the symptoms produced will aid in a clear understanding of the condensed tables given below. A typical case of rapid injury, such as is produced by a heavy application of Paris green at a point where a twig had just been cut off, runs the following course. In two or three days the cut surface of the wound has become darkened, and there is a slightly darker streak running upward and downward from this point. At either side of the wound the discoloration has extended only a short distance. By the end of a week

the discolored streak has widened and lengthened considerably, often reaching downward for a foot or so and upward to the end of the branch several feet away. Sometimes it forks and goes out on several limbs. It is now more definite in outline, somewhat sunken, and decidedly darker in color than the normal. The leaves on all twigs touched by this streak are more or less spotted. At first this spotting consists only of a loss of the normal lustre. The spots look dull and lifeless but are not discolored. Soon they become lighter and the tissue wilts and dies, either in spots or throughout the entire leaf. When dried up they are a light brown. Internally the discoloration is darkest and extends most rapidly in the cambium region, i. e., in the youngest phloem and xylem. It is a chocolate brown in the bark and a very dark reddish brown in the outer wood. In the vicinity of the wound this discoloration extends irregularly in the pith or beyond, though much lighter in color. A short distance away it is more superficial. In most cases studied the streaks do not continue to enlarge much longer than three weeks. Later they become more sunken, partly from drying, and partly from the growth of the surrounding tissues. They also become separated from the healthy bark by a sharp but usually slight crack.

Analyses made by Mr. Burke, the station chemist, showed the presence of arsenic in all parts of the treated branch—bark, wood and leaves even in the side branches that showed no visible symptoms. It was not found in the soil nor in an untreated tree.

In slower and less severe attacks there is only a dead and discolored spot of varying size about the wound. When lenticels are invaded the spots are at first superficial and the lenticel is usually near the centre. Later they penetrate to the cambium and, if numerous, they may spread and anastomose until the branch is quite girdled.

Observations were made at frequent intervals but those recorded in tables I to IV were about three weeks after the branches were treated, unless otherwise indicated. Table V represents the conditions fourteen weeks after the crowns were treated.

For the sake of convenience in studying the results, we will present the experiments in series. For reasons that we cannot enter into here, different amounts of the chemicals were sometimes used in the same series. This must be taken into account when comparing the results.

The results embodied in Table I suggest that any of the different chemicals used, excepting zinc arsenite, may do considerable damage to the limbs, but that the wounding of the trees is a very potent factor in determining the extent of this injury. The injury caused by zinc arsenite was so slight as to be almost negligible. Lenticels are shown to be points of attack.

TABLE I SERIES I ON BRANCHES (Preliminary)

No.	Kind of Chemical	Am't of Chemical	Variety of Tree	Diam. of Limb	Wounds under Bandage	Extent and Character of Injury.
1	Arsenic trioxide	60 g.	Gano	5/8 in.	Small pruning wounds of last two seasons.	Bark under bandage dead on under half where there are last spring's pruning wounds, and showing dead spots on upper half, each with lenticel in centre. Dead streak runs up 10 in. from bandage. Wood partly discolored. Leaves not injured.
2	Lead arsenate	150 g.	Wealthy	1 1/8 in.	Unwounded	Numerous dead spots under bandage, 1/8 in. or more in diameter, with lenticels in centres. Some of these anastomosing to form large dead patches.
3	Lead arsenate	150 g.	Transcendent crab	2 in.	Small pruning wounds of last spring	Dead spot under bandage 1 x6 in. connecting two wounds. Another spot 5 in. long and 3/4 to 1 1/2 in. wide connected with another wound. Smaller spots with lenticels in centres.
4	Lead arsenate	60 g.	Duchess	3/4 in.	One small twig just removed.	Dead streak 1/4 in. wide extends upward from wound 3 1/2 ft. to present season's growth, killing two twigs and turning leaves brown. Also several small spots under bandage with lenticels in centres.
5	Paris green	60 g.	Early Harvest	3/4 in.	1 sprout removed. 2 small pruning wounds of last spring.	Good sized dead spots around all three wounds. Several dead spots with lenticels in centres. These tend to run together.
6	Paris green	150 g.	Greenwood crab	1 3/4 in.	1 small limb and 1 twig removed. Many pruning wounds of different ages.	Branch cut off 4 days after treatment. Practically dead from bandage to tip (7 ft.) Dead streak runs 2 ft. down from bandage. Side branch 1 ft. below bandage nearly dead. Discoloration greatest around fresh wounds. Not especially prominent around wounds made 1 year or more ago.
7	Zinc arsenite	60 g.	Minnetonka	3/4 in.	Unwounded	One dead spot under edge of bandage 1/4 in. in diameter, with lenticel in centre.
8	Zinc arsenite	60 g.	Duchess	7/8 in.	1 twig removed	Two small lenticel spots under edge of bandage. Wound not effected.

TABLE II SERIES II ON BRANCHES

Showing the relative effects of different "insoluble" compounds and the part played by wounds in causing injury through the bark.

No.	Kind of Chemical	Am't of Chemical	Variety of Tree	Diam. of Limb	Wounds under Bandage	Extent and Character of Injury
1	Arsenic trioxide	5 g.	Hybernal	7/8 in.	Unwounded	Bark killed under bandage.
2	Arsenic trioxide	5 g.	Hybernal	7/8 in.	1 twig removed and tangential knife slash just to cambium	Wide streak of dead bark extends from wound downward 4 in. and upward to end of limb, killing tip and several small side branches.
3	Arsenic disulphide	10 g.	Hybernal	7/8 in.	Unwounded	One large and one small dead spot under bandage, with lenticels in centres.
4	Arsenic disulphide	10 g.	Hybernal	7/8 in.	1 limb 3/4 in. removed	Dead streak runs from wound downward 5 in. and upward 15 in. to a fork already dead.
5	Arsenic trisulphide	10 g.	Hybernal	3/4 in.	Unwounded	One dead spot under bandage 3/4 x 1 in. with lenticel in center.
6	Arsenic trisulphide	10 g.	Hybernal	7/8 in.	1 limb 1/2 in. removed	Bark on wounded side dead half way around from 3 in. below bandage to end of branch. One fork above bandage and several twigs entirely killed.
7	Calcium arsenite	10 g.	Hybernal	7/8 in.	Unwounded	Bark killed under bandage.
8	Calcium arsenite	10 g.	Hybernal	1 in.	2 twigs 3/8 in. removed	Bark killed under bandage, and a broad streak runs down 1 1/2 ft. and up to a fork, one portion of which is entirely killed.
9	Lead arsenate	60 g.	Okabena	3/4 in.	Unwounded	A little injury under bandage in a spot slightly scraped some time ago.
10	Lead arsenate	60 g.	Okabena	3/4 in.	1 small twig removed. 1 tangential knife slash to cambium	Two equal dead streaks run upward from the two wounds and fuse 1 ft. from bandage girdling the limb which is dead to the tip. Several small dead spots on bark under bandage. These do not contain lenticels.
11	Lead arsenate	4 g.	Hybernal	3/4 in.	Unwounded	No symptoms of injury.
12	Lead arsenate	4 g.	Hybernal	3/4 in.	2 twigs 3/8 in. removed	Dead spot 3/4 x 8 in. surrounding the two wounds.
13	Paris green	60 g.	Okabena	1 1/8 in.	Unwounded	Numerous dead spots under bandage, which have lenticels in centres. On the under side many of these are anastomosing.

TABLE II SERIES II ON BRANCHES (*Continued*)

No.	Kind of Chemical	Am't of chemical	Variety of Tree	Diam. of Limb	Wounds under Bandage	Extent and Character of Injury
14	Paris green	60 g.	Okabena	¾ in.	1 limb ⅜ in. removed. 1 tangential knife slash to cambium	Dead streaks run upward from the two wounds and fuse 7 in above the bandage. From here the streak runs continuously for 2½ ft. and then intermittently to end of branch. Several side branches are killed.
15	Zinc arsenite	60 g.	Okabena	¾ in.	Unwounded	No symptoms of injury.
16	Zinc arsenite	60 g.	Okabena	1⅛ in.	1 limb ¾ in. removed. 1 tangential knife slash to camb.	No symptoms of injury. Wounds healing normally in contact with chemical.

It may be seen from Table II that six of the chemicals used, namely, arsenic trioxide, arsenic disulphide, arsenic trisulphide, calcium arsenite, lead arsenate, and Paris green, are capable of causing considerable injury through openings in the bark but the zinc arsenite seems practically harmless even to the exposed surface of a fresh wound. Excepting in No. 10 there was no evidence of a direct absorption through the unbroken layer of cork protecting these limbs. In this case we must keep in mind the possibility of very tiny punctures through this thin layer. The chemicals may, however, enter and do serious damage through newly made wounds, through lenticels or at dormant buds.

The results shown in Table III indicate that any newly made wound that breaks the corky layer of the bark is sufficient to permit arsenical injury to the living cells inside. It is believed that had greater amounts of Paris green been used the differences in the injuries would have been much more striking. No. 7 is especially noteworthy as suggesting the effectiveness of paint even when fresh in preventing the absorption and resulting injury. Judging from No. 1 we might conclude that the absorption goes on through old healing wounds as well as fresh ones, but this conclusion is not supported by other observations made in the course of the investigations.

From Table IV it may be seen that sodium arsenite and arsenic acid are about equally injurious to the limbs, which is contrary to the widely accepted belief of Pfeffer[1] that arsenic acid is relatively harmless to plants. It has, of course, long been recognized that sodium arsenite is strongly toxic to foliage and to herbaceous plants.

It is of especial interest that both these compounds are injurious in

[1] Pfeffer, W. Physiology of Plants. 1: 438. 1900.

TABLE III SERIES III ON BRANCHES

Showing the relative injury through wounds of different size and character.

No.	Kind of Chemical	Am't of Chemical	Variety tree	Diam. of Limb	Wounds under bandage	Extent and Character of Injnry.
*1	Paris green	2 g.	Okabena	2 in.	Pruning wound of 1908	Dead spot 2 x 3½ in. around old pruning wound. No other injury.
2	Paris green	1 g.	Hybernal	¾ in.	1 twig removed	Dead spot 1 x 2 in. about wound.
3	Paris green	1 g.	Hybernal	⅞ in.	5 mm. sq. of bark removed half way to cambium	Dead spot ⅝ x ⅞ in. about wound. 1 spot around latent bud.
4	Paris green	1 g.	Hybernal	¾ in.	5 mm. sq. of outer cork removed	Dead spot ⅞ in. diam. about wound, 1 spot around latent bud and 1 lenticel spot.
5	Paris green	1 g.	Hybernal	⅞ in.	Longitudinal radial cut ¾ in. long to cambium	Dead spot ⅞ in. diam. about wound.
6	Paris green	1 g.	Hybernal	¾ in.	Transverse cut ¾ in. long to cambium	Dead spot extends 1¾ in. above wound, 1 in. below and ¼ in. on either side.
7	Paris green	1 g.	Hybernal	¾ in.	1 twig removed 1 tangential knife slash to cambium. Wounds painted with white lead and treated immediately	Slash wound shows no injury. Twig wound has very slight discoloration at lower margin of wound.
8	Paris green	⅕ g.	Hybernal	This season's shoot	1 leaf broken off	Dead spot ¼ in. diam. about wound.
9	Paris green	⅕ g.	Hybernal	This season's shoot	Unwounded	No symptoms of injury.
10	Paris green	⅖ g.	Hybernal	½ in.	Unwounded	No symptoms of injury.
11	Paris green	1 g.	Hybernal	1 in.	Unwounded	1 small lenticel spot.

*NOTE.—Direct comparisons cannot be made between this number and the rest of the of the series as, through an oversight, notes were not taken on this limb until 4 months after treatment.

TABLE IV SERIES IV ON BRANCHES

Showing the relative effects of two soluble compounds and the part played by wounds in causing injury through the bark.

No.	Kind of Chemical	Am't of Chemical	Variety of Tree	Diam. of Limb	Wounds under Bandage	Extent and Character of Injury.
1	Arsenic acid	200 cc. of 1-10 sol.	Green-wood crab	¾ in.	5 twigs removed	Branch removed at end of week. Entirely killed above bandage. Limb 4 in. below badly injured. Color darkest about wounds.
2	Arsenic acid	50 cc. of 1-100 sol.	Thompson No. 10	1 in.	Unwounded	Many dead spots under bandage with lenticels in centres. Some are anastomosing to form larger dead areas.
3	*Arsenic acid	50 cc. of 1-100 sol.	Hybernal	¾ in.	Small abrastions made last winter	Abont half the lenticels under bandage are centres of dead spots. Small dead spots around dormant buds and abrasions.
4	*Arsenic acid	50 cc. of 1-100 sol.	Hybernal	¾ in.	1 limb ½ in. removed	Nearly all above bandage killed. Below bandage wide streak runs down 15 in. killing several twigs.
5	*Arsenic acid	50 cc. of 1-1,000 sol.	Hybernal	¾ in.	Unwounded	A few of the lenticels and dormant buds under bandage are centres of dead spots.
6	*Arsenic acid	50 cc of 1-1,000 sol.	Hybernal	¾ in.	2 limbs ½ in., removed	Wide streak from one wound runs upward to the tip and nearly all above bandage is killed. Streak runs down from other wound for 1½ in.
7	*Arsenic acid	50 cc. of 1-100,000 sol.	Hybernal	¾ in.	Unwounded	Three tiny lenticel spots.
8	*Arsenic acid	50 cc. of 1-100,000 sol.	Hybernal	⅞ in.	1 twig removed 1 knife slash to cambium	Irregular dead spot around each wound. Bark under bandage a little darkened. Leaves show arsenical spotting.
9	*Arsenic acid	65 cc. of 1-1,000,000 sol.	Hybernal	¾ in.	Unwounded	No symptoms of injnry.
10	*Arsenic acid	55 cc. of 1-1,000,000 sol.	Hybernal	¾ in.	2 twigs removed	No symptoms of injury.
11	Sodium Arsenite	200 cc. of 1-10 sol.	Gano	⅞ in.	Unwounded	Branch cut off a week after treatment. All above bandage killed. Main branch from which treated one grew out considerably injured with spotted leaves.
12	Sodium Arsenite	50 cc. of 1-100 sol.	Okabena	1-⅛ in.	Unwounded	Many of the lenticels are centres of dead spots which are anastomosing.
13	*Sodium Arsenite	50 cc. of 1-100 sol.	Hybernal	⅞ in.	Unwounded	Dormant buds under bandage are centres of dead spots.

NOTE.—In these cases the bandages were wrapped with dental rubber dam.

TABLE IV SERIES IV ON BRANCHES (*Continued*)

No.	Kind of Chemical	Am't of Chemical	Variety of tree	Diam. of Limb	Wounds under Bandage	Extent and Character of Injury
14	*Sodium arsenite	60 cc. of 1-100 sol.	Hybernal	¾ in.	2 twigs ⅜ in. removed	Entire branch above bandage killed. Below bandage two streaks run down 2½ ft. in parallel spirals from the two wounds. All bark under bandage dead.
15	*Sodium arsenite	40 cc. of 1-1,000 sol.	Hybernal	⅝ in.	Unwounded	A few lenticels under bandage are centres of small dead spots.
16	*Sodium arsenite	40 cc. of 1-1,000 sol.	Hybernal	⅝ in.	1 twig ¼ in. removed	Streak from wound runs up for 22 in. and kills twig and side branch.
17	*Sodium arsenite	40 cc. of 1-100,000 sol.	Hybernal	⅞ in.	Unwounded	Several tiny lenticel spots.
18	*Sodium arsenite	60 cc. of 1-100,000 sol.	Hybernal	¾ in.	2 twigs removed	Streaks run up and down a few inches from wounds. Some of the leaves show arsenical spotting.
19	*Sodium arsenite	50 cc. of 1-1,000,000 sol.	Hybernal	⅝ in.	Unwounded	No symptoms of injury.
20	*Sodium arsenite	50 cc. of 1-1,000,000 sol.	Hybernal	¾ in.	1 twig removed	No symptoms of injury.

*NOTE.—In these cases the bandages were wrapped with dental rubber dam.

very high dilutions, i. e. 1–100,000, even to the bark of apple trees, which emphasizes the importance of having the arsenical insecticides free from these impurities. It also calls our attention to the danger that arises when alkali salts change portions of these chemicals that run to the ground into sodium arsenite. It should be kept in mind that in the higher dilutions and most of the others, a concentration of the solutions in the bandages through evaporation was prevented by wrapping in sheet rubber.

Table V shows that the same treatment as that already described, when given to the crowns, causes, as might be expected, similar injuries. No observations have yet been made on the symptoms below ground as it is desired to leave the chemicals undisturbed about the crowns through the present winter at least.

Considering the results of the experiments as a whole, we find little room to question that the arsenicals commonly used as insecticides are capable of doing serious injury through the bark under certain conditions. Whether this injury is really caused by the chemicals named *per se* or by soluble arsenical impurities we hold open to serious question. As has been stated only high grade chemicals were used and yet we were led to suspect that, in some cases at least, the injury was due to soluble impurities.

TABLE V SERIES V ON CROWNS

Showing relative injury to crowns wounded and unwounded, by different insoluble compounds.

No.	Kind of Chemical	Am't of Chemical	Variety of Tree	Size of Trunk	Wounds at Crown	Extent and Character of Injury.
1	Arsenic trioxide	7 g.	?	4½ in.	Unwounded	No symptoms above grounnd.
2	Arsenic trioxide	7 g.	?	4¾ in.	Strips of bark ¾x1½ in. removed, 1 from N. and 1 from S. side just below ground	On north side dead streak 1 to ¼ in. wide runs from ground up to branches, 2½ ft.
3	Arsenic trisulphide	15 g.	?	4¾ in.	Unwounded	No symptoms of injury above ground.
4	Arsenic trisulphide	15 g.	?	4¼ in.	Same as No. 2	On north side dead streak 1¼ in. and growing narrower runs from ground up trunk (2 ft.) and out on branch, 1 ft. more.
5	Lead arsenate	60 g.	?	2 in.	Unwounded	No symptoms of injury above ground.
6	Lead arsenate	60 g.	Salome	4½ in.	Water sprouts cut off all around. A few old healing wounds	No symptoms of injury above ground.
7	Paris green	15 g.	Royal Table	3¼ in.	Unwounded	No symptoms of injury above ground.
8	Paris green	15 g.	Okabena	3 in.	Water sprouts cut off all around the crown	On south side dead patch half around the trunk at ground line. From this a streak runs up 1 ft., disappears for 10 in. (*i.e.* discolors the inner bark only) and reappears as a streak ½ in. x 3 ft. on south fork. On north fork another streak ½ in. x 20 in. Leaves on twigs touch by streaks badly injured, and cling after others have fallen.
9	Zinc arsenite	15 g.	Royal Table	4¼ in.	Unwounded	No symptoms of injury above ground.
10	Zinc arsenite	15 g.	Gano	4 in.	Same as No. 2.	No symptoms above ground at the end of 3 weeks when the tree was broken down by storm and removed.

TABLE IV

Number of cells in corky layer of smooth bark, and in the furrows of rough bark

Year's growth	Variety	Diameter	Character of bark	Cells in cork layer	Variety	Diameter	Character of bark	Cells in cork layer	Variety	Diameter	Character of bark	Cells in cork layer
1910	Wealthy	3/16 in.	Smooth	2-5	Hybernal	3/16 in.	Smooth	1-3	Okabena	1/4 in.	Smooth	3-5
1909	"	3/16 "	"	2-5	"	5/16 "	"	4-6	"	1/4 "	"	2-5
1908	"	1/4 "	"	2-5	"	3/8 "	"	3-7	"	5/16 "	"	3-5
1907	"	3/8 "	"	3-7	"	7/16 "	"	3-7	"	3/8 "	"	4-6
1906	"	3/8 "	"	2-7	"	1/2 "	"	4-7	"	3/8 "	"	4-8
1905	"	5/8 "	"	4-8	"	3/4 "	"	3-7	"	5/8 "	"	4-9
1904	"	7/8 "	"	3-7	"	7/8 "	"	2-8	"	3/4 "	"	4-8
1903					"	1-1/8 "	"	5-7				
1902					"	1-3/8 "	"	1-8	Okabena	7/8 "	"	3-8
1900									"	1-1/8 "	"	4-10
1889									"	1-1/4 "	"	4-9
1896									"	2-1/4 "	"	4-9
	Wealthy	4 in.	Smooth	3-many	Liebe	4 in.	Smooth	4-many	"	4 "	"	4-many
	Duchess	6 "	Rough	6-many	"	6 "	Rough	4-many	Trans. crab	6 "	Rough	2-many
	Orange	8 "	"	3-many					"	8 "	"	5-many
									"	12 "	"	1-many

It will be noted that the results of these experiments are radically different from those obtained by Ball, Titus and Greaves[1] who treated limbs of trees with different strengths of lead arsenate, "Kedzie arsenate," Paris green and sodium arsenate, but obtained no injurious results other than a slight burning of the leaves in some cases. No injury to the bark was reported, and naturally the part played by wounds, lenticels, and dormant buds did not appear. They did not, however, describe their methods nor state the exact amounts used; and the difference in results may be due to applying the chemicals in a different way from that which was used at this station. The corky tissue forming the outer layer of the smooth bark seems to prevent almost completely the absorption of the chemicals. As may be seen from Table VI this layer is from 1 to 10 cells in thickness. The age of the bark makes very little difference in the thickness of the cork up to the time when the surface becomes rough. As the outer cells slough off, new ones form within to take their place. The bottom of some lenticels are well protected by a compact corky layer while in others this layer is totally lacking. Both conditions may be found close together on limbs varying at least from ½ inch to 1¼ inches in diameter. Whether or not the rough bark on the older portions offers a perfect protection remains to be determined by further investigations. In this older bark the outer layers are being ruptured by growth from within and in the deepest furrows the cork is sometimes as thin as on branches having a smooth surface. We do not, however, consider this microscropical evidence sufficient in itself to prove which would be the better protection against the absorption of arsenical solutions. It certainly is not proven by these experiments that arsenicals can not penetrate this corky layer and do harm, if allowed to remain in contact with the expanding rough bark of the crown through a considerable period of years.

The most striking thing shown by these tests is the part played by wounds in permitting the absorption of the arsenical solutions. As a rule there was little injury through wounds made a year or more before the arsenical treatment, but those made at the time the chemicals were applied or at the end of the previous dormant season let the solution enter freely, unless painted as was done in one case.

Attention has already been called to the fact that as a rule the injury did not increase after a few weeks. This is taken to indicate one of two things; either that the available arsenical solution was absorbed and deposited in the tree in some form that did not readily pass to the

[1] Ball, M. D., Titus, E. G. and Greaves, J. E. The Season's Work on Arsenical Poisoning of Fruit Trees. Jour. Econ. Ent. 3: 187. 1910.

extremities, or that the injured bark and wood are so changed as no longer to conduct the solutions from the supply in the bandages. That the latter is the correct explanation is indicated by the fact that the action of sodium arsenite and arsenic acid in weak solutions was checked while there was still an abundance of the solution in the bandages under the rubber.

Lastly, it may be noted that the arsenic trisulphide tenatively suggested by the Colorado station has not proved less harmful in these tests than Paris green, calcium arsenate or lead arsenate. That this compound is relatively insoluble in pure water is an accepted fact; but when brought in contact with the juices of the tree at the wounds it appeared to be changed into a soluble form and absorbed freely, with serious effects.

CONCLUSIONS

From these experiments we may draw the following conclusions, subject to verification by further tests.

1. Serious injury to apple trees may, under certain conditions, result from applications of the so-called "insoluble" arsenical insecticides.

2. Among the conditions that favor this injury, recently made wounds through the outer bark are highly important.

3. Functional lenticels and dormant buds also permit the absorption of arsenical compounds in solution.

4. The corky bark is highly protective and does not readily become disintegrated by the action of arsenical compounds.

5. It is not safe for orchardists to cut off water sprouts from the bases of trees in early spring and permit the arsenicals now used as insecticides to accumulate about the unprotected wounds the same season.

6. White lead paint applied to these wounds will practically protect them even if put on just before the arsenicals are applied.

7. The most noticeable symptoms of rapid arsenical injury through the bark are a discoloration of bark and outer wood, often following definite lines up and down the stem, and a dull spotting of the leaves followed by wilting, shriveling, and drying.

8. It is not improbable that the injury is due more to soluble impurities than to the slight solubility of the compounds themselves.

9. If such should prove the case, the remedy for the alleged arsenical injury to crowns (excepting perhaps in alkali soils) lies not in discontinuing the use of arsenical insecticides but in securing grades of higher purity.

10. Arsenic trisulphide does not promise to be more safe when ap-

PLATE XVI **Arsenical Injury**

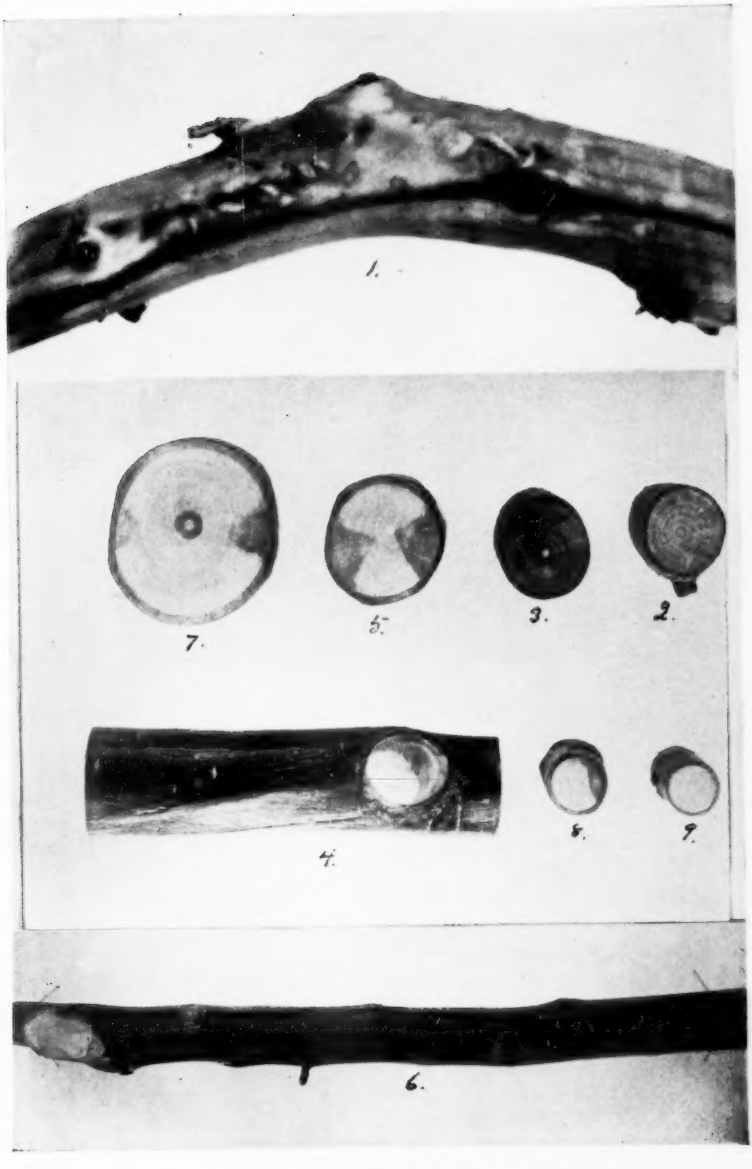

PLATE XVII **Arsenical Injury**

plied to the bodies of trees than the arsenical insecticides now in common use.

11. Zinc arsenite gave practically no injury under the most severe conditions of the tests. If this compound bears up as well during future tests and proves a satisfactory insecticide, it may become generally recommended, especially if it does not prove practicable to get grades of Paris green and lead arsenate that will not injure through wounds.

MONTANA EXPERIMENT STATION,
BOZEMAN, MONTANA

Arsenical Injury.

PLATE XVI shows bandages on treated limbs, and boxes for preventing the solutions from dripping to the ground.

PLATE XVII shows character of arsenical injury.

FIG. 1. Limbs of Greenwood tree treated with a solution of arsenic acid. The portion photographed was enclosed by the bandage. Bark removed for photograph. The knot in the dark injured streak was made by cutting off a limb when the solution was applied. The other knots shown in the figure were old and showed no arsenical injury. Taken natural size.

FIGS. 2 to 9. Portions of Hybernal tree injured by a single treatment with sodium arsenite solution. There were two fresh wounds under the bandage on opposite sides of limb.

FIG. 2. Cross section 1 foot above bandage. Natural size.

FIG. 3. Cross section under bandage just below wounds. Natural size.

FIG. 4. Surface view 8-11 inches below bandage. Natural size.

FIG. 5. Cross section 16 inches below bandage, just above main limb. Natural size.

FIG. 6. Main limb with treated limb removed (wound made by its removal shown near left end of figure). About ¼ natural size.

FIG. 7. Cross section of main limb 1 foot below intersection with treated limb.

FIGS. 8 and 9. Cross sections of side branch that joined treated limb 8 inches below bandage (wound made by its removal shown in Fig. 4.) Figure 8 is one inch and figure 9 is six inches above intersection with treated limb. Natural size.

The injuries here shown are very similar to those caused by Paris green, calcium arsenite, arsenic trioxide, etc.

(All slightly reduced in reproduction.)

THE RUSTS OF TSUGA CANADENSIS[1]

PERLEY SPAULDING

(WITH TWO MAPS IN THE TEXT)

The rusts of *Tsuga canadensis* (Linn.) Carr. have been collected by the writer for several years in various localities in New York and New England. Besides this material, some was collected in North Carolina during the past summer by Dr. A. H. Graves on both *Tsuga canadensis* and *T. caroliniana* Engelm. In 1908 when the writer first began these collections and observations the following species of rusts were known to attack *Tsuga canadensis*: *Peridermium peckii* von Thümen on the leaves, *Necium farlowii* Arthur on the leaves, and *Caeoma abietis-canadensis* Farlow, also on the leaves The first collection made by the writer was a rust upon the green cones and young shoots. None of the collections have proved to be *Necium farlowii*, altho special attention has been given to the search for this species.

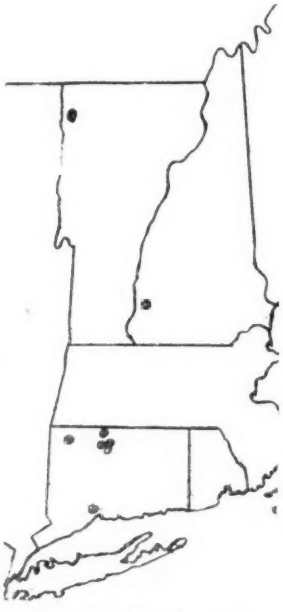

FIG. 1. Map showing the present known distribution of *Peridermium fructigenum* Arthur.

The larger number of the collections were *Peridermium peckii*. This is apparently the most abundant and widely distributed of the Tsuga rusts. It has been previously reported from various localities in New Hampshire, Vermont, Massachusetts, Connecticut, New York, Virginia, West Virginia and Wisconsin, upon *Tsuga canadensis* only. Graves collected it upon *T. canadensis* and also *T. caroliniana* in North Carolina. The writer's collections of this species were made at New Haven, Granby, East Granby, and Bloomfield, Connecticut, and Santa Clara and Essex, New York. These localities merely extend the known range into northern New York. Judging from the present known localities of occurrence, it is likely to be found throughout the range of the two eastern species of Tsuga. See Figure 1 for its known distribution and the range of its two hosts. Whether it may also occur on the western hemlocks is not known, but it has never been reported thereon.

The remainder of the material was easily separated into two forms: one on the scales of the green cones, and the other on the young, newly-formed shoots, of *T. canadensis* only. Arthur has recently de-

[1] Published by permission of the Secretary of Agriculture.

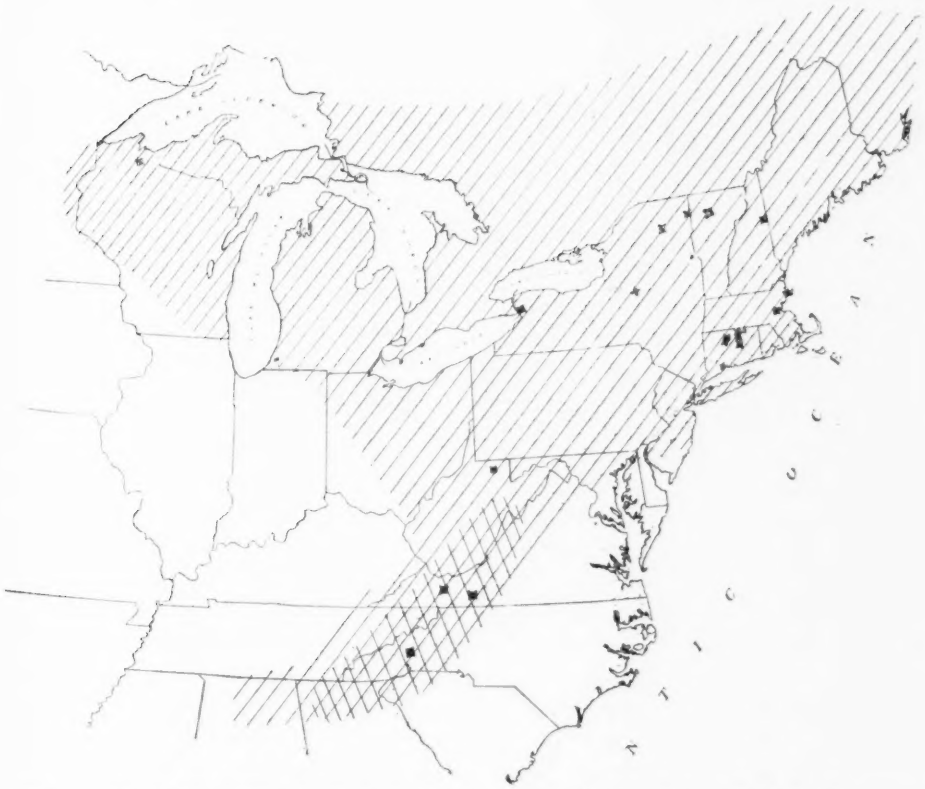

FIG. 2. Spotted localities show where *Peridermium peckii* has been collected : the cross-hatching shows the distribution of the two host species.

scribed and named the cone rust, *Peridermium fructigenum*.[1] The writer, unaware of the new species of Arthur, proposed to call it *Caeoma tsugae*,[2] and did not learn of Arthur's species in time to prevent this name appearing in print. Besides the original locality cited by Arthur for this fungus, the writer has collected it as follows : East Granby, Connecticut, June 26, 1909, and July 1, 1910 ; Bloomfield, Connecticut, June 21, 1909, and July 4, 1910 ; Granby, Connecticut, June 26, 1909 ; Simsbury, Connecticut, July 11, 1909 ; New Haven, Connecticut, June 20, 1909 ; Marlow, New Hampshire, July 9, 1909 ; Essex, New York, July 6, 1909 ; and Burlington, Vermont, July 7, 1909. See Figure 2 for the present known distribution of *Peridermium fructigenum*. The rust on the young shoots corresponded with that on the cones in the characters of the spores and aecidia. No specific

[1] Arthur, J. C. New species of Uredineae—VII. Bull. Torrey Bot. Club. **37**: 578. 1910.

[2] Spaulding, Perley. Rusts of Tsuga. Science, n. s. **33**: 194. 1911.

difference could be detected ; however, fresh material is needed to make sure that the two forms are identical. For the present they are considered to be the same species by the writer. This form has been collected only on *Tsuga canadensis* shoots at East Granby, Connecticut, June 26, 1909, and Granby, Connecticut, June 26, 1909.

The occurrence of these fungi in the various localities where found was interesting. *Peridermium peckii* occurred in many places where the others did not, and also where they did. Apparently there was no significance to be attached to its occurrence with the other two. The cone form of *Peridermium fructigenum* was found in a number of places where the twig form was not found, but the latter was found in the same localities for three consecutive years. There has been marked variation in the occurrence of the two forms of *Peridermium fructigenum*. In 1908 many trees having rusted cones also had the twigs diseased, but this was not true in all cases. In 1909 there was much less of the latter form, while the rusted cones were about as plentiful as they were the year before. But in 1910 the rusted cones were much less numerous, and the twigs were not at all affected. On the other hand, in Bloomfield, Connecticut, the cone form only has been found upon certain trees.

The economic importance of the various hemlock rusts is slight, as they have in no case yet seen caused anywhere near a complete infection of all the leaves, cones, or shoots of a single tree. The cone fungus is of greatest economic importance, since it prevents the production of seed in the diseased cones. However, no case has yet been seen where over fifty per cent. of all the cones were affected even on small trees. Dr. Arthur's suggestion that *Pucciniastrum minimum* (Schw.) Arthur on *Azalea* spp. is probably the alternate stage of *Peridermium fructigenum*, the writer believes will prove to be true. But if such is the case, the distribution of *Pucciniastrum minimum* is far from being definitely known at present.

Peridermium fructigenum fruits in the latter part of June, and is quite conspicuous as the bright golden-yellow rusted cones contrast sharply with the green foliage and healthy cones. This has never been found in the more elevated parts of central Vermont nor in the Adirondack region, though special search has been made in the proper season. This seems to indicate that the alternate host does not occur in these parts, but does occur in the warmer parts of New England, which is the case with the hosts of *Pucciniastrum minimum*.

INVESTIGATIONS IN FOREST PATHOLOGY,
BUREAU OF PLANT INDUSTRY,
WASHINGTON, D. C.

IS BACILLUS COLI EVER A PLANT PARASITE?

JOHN R. JOHNSTON

Although the organism known as *Bacillus coli* has been found in a great variety of places, it has never, so far as has been ascertained, been reported as causing a specific disease of plants. Several investigators have tried inoculating this organism into various plants but without success, except when tubers of potatoes, etc., were put under abnormal conditions (Laurent). The following is a brief summary of some inoculations carried out by the writer.

A. Inoculation into the heart tissues of a coconut seedling about one meter high in a greenhouse in Washington, Feb. 17 to Mar. 7, 1910. Result, a small watersoaked area about the inoculation hole.

B. Same condition, Feb. 17 to Apr. 5, 1910. Result, watersoaked and soft-rotted area 5 cm. long.

C. Same conditions, Apr. 14 to May 16, 1910. Results: No. 1, small extent of watersoaked and rotted area about inoculation hole; No. 2, soft rot extending 5 cm.

D. Same conditions, May 7 to June 8. Results: No. 1, watersoaked area 2.5 cm.; No. 2, slight watersoaked area; No. 3, watersoaked area 2.5 cm. long; No. 4, watersoaked area 1.5 cm. long; No. 5, watersoaked area 3 cm. long; No. 6, watersoaked area 8 mm. long; No. 7, slight watersoaked area; Nos. 8, 9, 10, checks showing no watersoaked area.

E. Inoculations into three trees in Cuba, Aug. 15 to Sept. 28. All three inoculations infected the trees and produced brown, wet-rotted areas.

F. Inoculations into coconut seedlings in greenhouse, Oct. 14 to Nov. 10. Ten inoculations. Six showed only a slight browning of the tissues; two showed good brown rots 3 cm. long; one showed a typical soft wet rot 3 cm. long and a brown stain 5 cm. long; and one showed a wet rot 12 cm. long.

B. coli was recovered from inoculation A, from B, from Nos. 1 and 2 of C, from Nos. 1 and 3 of D, from all three of E, but from F no platings were made.

It will be seen from these experiments that in the case of some inoculations soft rots were obtained and in others no distinct rot resulted. In all cases at least a small amount of brown watersoaked tissues were the result whereas in checks absolutely no discoloration resulted more than 1 or 2 mm. from the hole. The importance of the failure to secure results in every case is minimized by the difficulty of making the inoculations in the centre of soft tissues at the growing point. Soft rots resulted in these tissues except when the inoculation passed above or below or to one side, in the hardened tissues. Here it seldomed happened. It should be noted that the above inoculations were made with cultures of *B. coli* of animal origin obtained from different sources; inoculations A, B, and C were with subcultures from

a culture obtained from the Bureau of Animal Industry; D was made with two different cultures that had been used in the Pathological Laboratory of the Bureau of Plant Industry for two years or more; and the inoculations under E and F were made with a culture obtained directly from Dr. Theobald Smith. The identification of *B. coli* was in each case based as follows: upon the appearance of the colonies on Dolt's litmus-lactose-glycerin agar plates; upon the reaction in litmus milk of pink colonies from the plates; upon the reduction of nitrates in nitrate bouillon by transfers from those milk tubes that had reddened the litmus and coagulated the milk; upon the reaction in fermentation tubes containing peptone, dextrose and neutral red of transfers from those nitrate bouillon tubes that showed reduction of the nitrates; upon the reaction in gelatin of transfers from those fermentation tubes that showed from 25 to 75% gas production and a change of the red to a lemon-yellow color; and upon the production of indol in Dunham's solution of transfers from those gelatin tubes that showed liquefaction.

The stem tissues of the coconut seedlings are very hardy and in no case did any of these inoculations actually kill the plant. In several cases, however, the internal tissues were so rotted that subsequent death and dying out of the plant would have resulted. These experiments with the exception of E were carried on in a greenhouse on the grounds of the U. S. Dept. of Agriculture and with coconut seedlings and in consequence have relatively little economic value in themselves. What lends interest to these results is summarized in the following paragraphs.

At Baracoa, Cuba, there were isolated on August 7, 1909, from two different coconut trees seriously affected with the Budrot disease, cultures of bacterial organisms which were inoculated into seven apparently healthy trees. The inoculations were examined on days from Aug. 24 to Aug. 26 and in each case a soft white rot typical of the Budrot disease had resulted. The extent of the rot induced by the inoculations varied from 5 cm. to 60 cm. Isolations were made from these artificially diseased trees and cultures were taken to Washington for study. Four of these from as many different trees were inoculated from Sept. 24 to Sept. 29 into coconut seedlings in the greenhouses and in each case typical soft rots or at least watersoaked areas resulted. Bacteria were isolated from Oct. 16 to Oct. 27 from these rotted tissues and then comparisons were made with the original cultures. By a long series of cultural studies it was ascertained that at least two of the cultures which were originally from the same tree were identical; inoculation with these two into different trees each produced typical soft rots; isolation from these two trees gave exactly the same organisms as were inoculated into

them and also the same as were inoculated into the first artificially infected trees. It was found, too, that many of the other cultures were similar but only in the case of these two series was the comparison carried out to any extent.

As a result of these inoculations and the subsequent cultural studies it seemed conclusive that at least one organism capable of causing the Budrot of the coconut palm had been identified. Many earlier inoculations and incomplete cultural studies tended to corroborate this belief. It was found on examining the cultural characters of the coconut organism that they corresponded to the cultural characters of *B. coli*. For this reason the principle means of identifying *B. coli* were applied to the organism isolated from the diseased coconut trees. Cultures of *B. coli* of animal origin were studied in comparison with the coconut organism. This work was carried out in great detail and it was found impossible to ascertain any characters by which the coconut organism could be distinguished from the *B. coli*, received from the animal pathological laboratories. Finally the *B. coli* cultures of animal origin themselves were inoculated into the coconut plants as has already been mentioned. While the inoculations were not in all cases satisfactory yet there appears sufficient evidence to justify the conclusion that *B. coli* can destroy the soft tissues of the coconut plant, and that either *B. coli* or a form indistinguishable from it by our present methods of research is the cause of the coconut Budrot.

The work involved in making inoculations in the coconut palm is very great and the conditions are much complicated. For this reason these studies have been long drawn out and the utmost care has been taken to ascertain all factors involved in the problem. The foregoing paragraphs are a bare summary of the results and are not in themselves sufficient to judge the accuracy of the conclusions. They are presented here that they may be a suggestion to other workers on the coconut palm disease and also to call attention to the apparent production of a plant disease by *B. coli*. The presence of very closely allied forms in the many cultures made by the writer together with the presence of a form indistinguishable from *B. coli* indicate a probable greater importance of this organism in relation to the vegetable world than has heretofore been recognized. The work of the writer on this subject which was carried out for the Laboratory of Plant Pathology, U. S. Department of Agriculture, has been concluded and the data together with the discussion is now in preparation for the printer, as a Bulletin of the Bureau of Plant Industry.

RIO PIEDRAS,
PORTO RICO

PARASITISM OF CONIOTHYRIUM FUCKELII

P. J. O'GARA

(A PRELIMINARY PAPER WITH PLATES XVIII—XXI)

In October, 1903, specimens of apple twigs and young apple trees, taken from a nursery, were sent to the writer at Washington, D. C., by Dr. Haven Metcalf, Plant Pathologist and Bacteriologist, then at Clemson College, South Carolina. Upon examination, these specimens showed cankers of various sizes, the surface of which bore numerous minute pycnidia. The young apple trees were infected mostly at a point not very far above the crown. In young cases the bark tissue still adhered to the wood, but in the older infections the dead bark had broken away. A careful microscopical examination showed that the organism, which apparently caused the cankers, was a species of *Coniothyrium*, and subsquent work proved it to be *Coniothyrium fuckelii* Sacc. The description of the organism is as follows :

Perithecia situated beneath the epidermis, scattered, black, 180-200μ in diameter, globose-depressed ; ostiole barely prominent ; spores very numerous, globular or short elliptic, $2-4.5\times2-3.5\mu$, olivaceous or slightly sooty colored, basidia not visible or only slightly so.

Pure cultures made by the poured-plate method, were easily obtained, and the fungus was grown in various culture media, including apple agar, beef agar, potato agar, steamed rice, potato tubes and apple tubes. The organism fruited readily, producing typical pycnidia and spores varying somewhat in size, depending upon the medium but all within the limits of the species. In these cultures the mycelium consisted of very thin, branching, septate hyphae at first hyaline and later somewhat dark or fuscous. In very old cultures the mycelium became almost black, or a dark brown. The number of spores found in a nature pycnidium averaged nearly 300,000, varying somewhat, depending upon the medium in which the organism was grown.

About the same time that the specimens of apple twigs and trees were received from Dr. Metcalf, the writer found several specimens of *Rosa* sp. in the Congressional Cemetery at Washington, D. C., affected with cankers which, upon examination, proved to be very much like those found upon the apple. An examination of the pycnidia showed a close similarity to the organism found upon the apple, and subsequent growth in pure cultures proved the identity of the two organisms so far as morphological and cultural characters were concerned.

During the summer of 1904, inoculation experiments were undertaken, and from pure cultures, taken from the apple and rose, artificial infections were produced on several species of rose, and on several varieties of apples, including some Russian varieties growing on the Potomac Flats. Cross-inoculations made from pure cultures readily produced cankers on both the rose and the apple, no difference having been noted in any case. Inoculations were also made on the apple fruit from pure cultures originally taken from rose and apple, and again no difference was noted in the rot which was subsequently produced. The cankers produced on the rose by the organism taken from the apple were typical in every respect, and the morphological characters of the organism were identical with those of the organism originally found on the rose. Likewise, the same was true of the rose organism when inoculated into the apple. The inoculation experiments were all carried on in the field under normal conditions. Infections were produced very easily in roses and apples, both fruit and twigs, when ripe spores or mycelium were used for inoculation. The cankers produced were exactly the same in every case, and from these cankers the organism was readily obtained and proved to be identical when grown in pure cultures. The experiments were repeated a number of times so that, in all, no less than 300 inoculations were made.

The rot upon the fruit is quite typical producing a circular, brownish and somewhat sunken area which is smooth at first and later somewhat wrinkled, due to evaporation from the browned and rotted tissues beneath. The mycelium, which may be seen in the rotting tissue, is at first densely white and later becomes brown. The disease, when it attacks the fruit of the apple, often rots it completely.

The manner of natural infection has not been fully determined, but it was quite noticable in a nursery near Clemson College, South Carolina, that most of the infections took place where the bark of the trees had been bruised or slightly broken by the tools or harness in cultivating. In another nursery, serious infection was found on apple trees which adjoined a clump of wild roses badly infected with the disease. The trees nearest the roses showed the greatest number of infections. No detailed study has been made as to the susceptibility of the different varieties of apples, but it has been observed that some varieties show considerable immunity. The Newman apple, which is a local variety grown near Clemson College, seemed more susceptible, and it was upon this variety that the disease was first found.

As soon as possible more complete studies will be made and the results published.

This disease was observed by F. L. Stevens and J. G. Hall, in 1906, in North Carolina, and some of the results were published in Bulletin No. 196, bearing the date of June 1907, of the North Carolina Agricultural Experiment Station. The cankers on the twigs and the rot of the fruit were noted by them, but no work was done to show the relationship existing between the disease on roses and apples.

The accompanying photographs show cultures of the organism and natural and artificial infections on apples and roses.

MEDFORD, OREGON

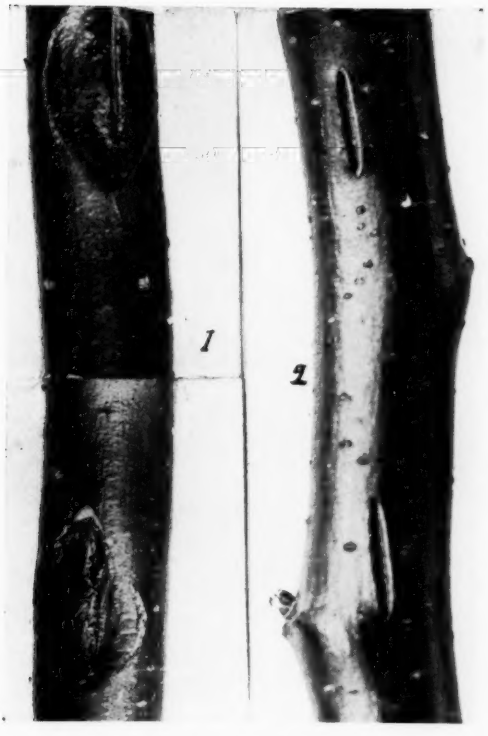

PLATE XVIII. **Coniothyrium Canker of Rose and Apple**

FIG. 1. Artificial infection of apple with *Coniothyrium fuckelii* taken from rose, x 2. (1904)

FIG. 2. Check showing stab with sterile scalpel, x 2. (1904)

PLATE XIX. **Coniothyrium Canker of Rose and Apple**

FIG. 1. Artificial infection of rose with pure culture of *Coniothyrium fuckelii* taken from apple. (1904)

FIG. 2. Natural infection on rose. (1904)

FIG. 3. Artificial infection of apple with pure culture of *Coniothyrium fuckelii* taken from rose. (1904)

FIG. 4. Check showing apple twig stabbed with sterile scalpel. (1904)

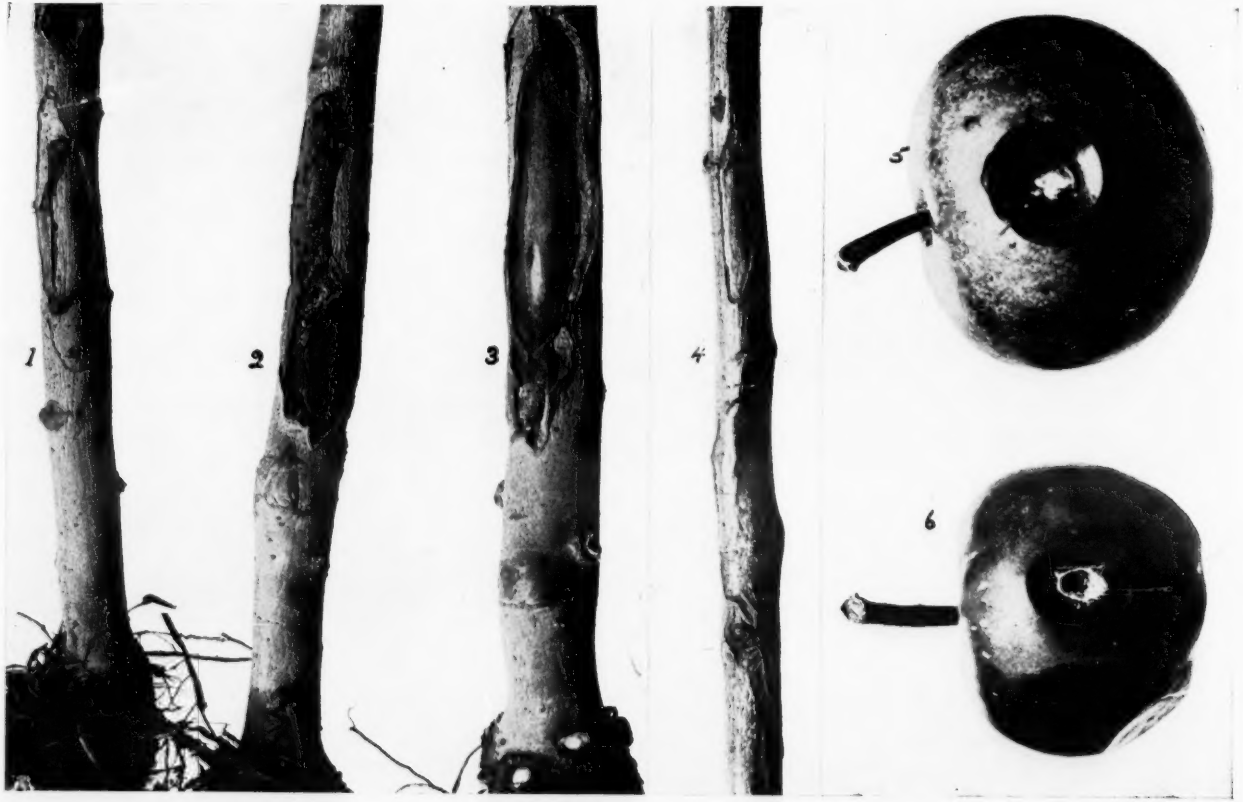

PLATE XX. **Coniothyrium Canker of Rose and Apple**

FIG. 1, 2, 3. Natural infections on nursery stock grown near infected roses. The injured bark has dropped out making an open canker. (1903)

FIG. 4. Natural infection on twig of four year old apple. From this specimen pure cultures were made for subsequent artificials inoculations. (1903)

FIG. 5. Artificial infection of apple with pure culture taken from rose. (1903)

FIG. 6. Artificial infection of apple with pure culture taken from apple. (1903)

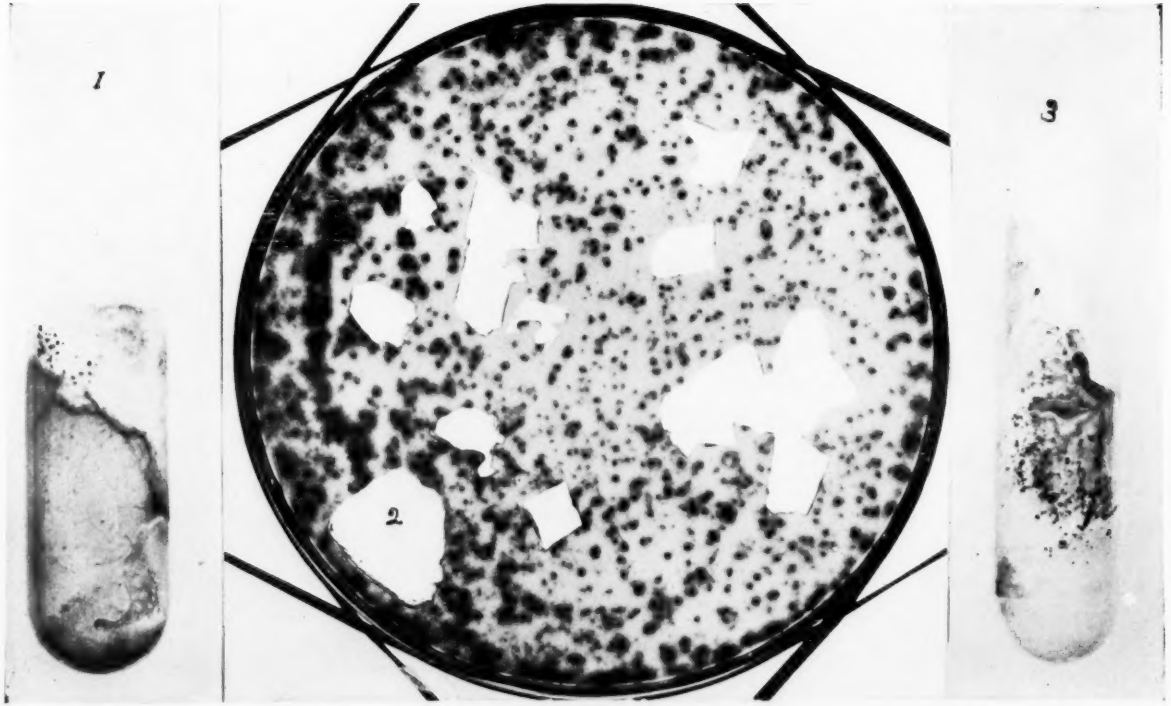

PLATE XXI. **Pure Cultures of Coniothyrium fuckelii from Rose and Apple**

FIG. 1. Pure culture of *Coniothyrium fuckelii* growing on potato slant, taken from infected rose. (1904)
FIG. 2. Pure culture of *Coniothyrium fuckelii* on neutral beef agar. Petri culture ten days old. (1904)
FIG. 3. Pure culture of *Coniothyrium fuckelii* taken from apple. Growing on sterile potato slant. (1904.)

RHIZOCTONIA MEDICAGINIS IN AMERICA[1]

F. D. HEALD

In the recent work by Duggar on "Fungous Diseases of Plants" the statement is made that *Rhizoctonia Medicaginis* DeC. (*R. violacea* Tul.) is not known in this country, and Stevens and Hall in their "Diseases of Economic Plants" give the somewhat indefinite information that it has not yet appeared in many parts of America. Freeman[2] mentions the disease as occurring in Kansas and states that "it is now rapidly spreading in the alfalfa fields in this state." The disease was first reported in this country, by the writer from Nebraska[3] and the following is taken from some unpublished notes concerning observations made on this trouble near Columbus, Nebraska.

The affected field was six years old and located on a rather light sandy loam in the Platte Valley. The soil was tested and found to have a distinct acid reaction. The spots affected by the fungus were slightly irregular or circular in outline and varied in diameter from eight feet to three rods or more. The great majority of the plants within the affected patches were dead, while those which had survived were not vigorous and had always lost the main tap root. Sometimes the tap root was completely rotted, and crumbled in fragments when the soil was disturbed. In less affected plants the cortex of the roots would slip off easily when the plants were lifted from the soil, leaving the central woody cylinder. Plants within the affected patches which had survived were supported by a few small lateral roots which originated from the crown. Strands of the violet or brown mycelium were found abundantly in the soil and all roots not completely rotted showed the characteristic dense mantle of violet-colored hyphae. In a few cases the fungous mantle extended up around the base of the stems. As already stated, some of the patches had a definite circular outline. In these the center was occupied by a blue grass sod, while around this was a ring of dead plants, then a zone of plants showing little evidence of life, and still farther out the zone of slight attack. Slightly affected plants showed more or less yellowing of the foliage, but in many cases the leaves would wilt and die while still green. The fungus did not spread very rapidly as the owner reported that the affected patches had increased only 3-6 feet radially during the preceeding season.

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[1] It is interesting to note in this connection that Massee (Dis. Cult. Pl. and Trees: 236) allies *Rhizoctonia* with *Rosellinia*, rather than *Corticium*. He also assumes that the alfalfa *Rhizoctonia* and the potato *Rhizoctonia* belong to the same species but probably no American pathologists who have worked with both forms will accept this idea. He further fails to accept Rolf's work and denies any connection between *Rhizoctonia* and the basidium fruit of *Corticium vagum* Berk.& Curt. var. *Solani* Burt He refers this basidiomycete to *Hypochnus solani* Prill. & Del.

[2] Freeman, E. M., Bull. Kan. Agr. Exp. Sta. 155 : 322. 1908.

[3] Heald, F. D., Ann. Rep. Nebr. Agr. Exp. Sta. 19 : 40. 1906.

REVIEWS

In a series of articles in Naturwissenchaftliche Zeitschrift für Forst-und Landwirtschaft, Dr. Ernst Münch of the Forestry Experimental Institute at Munich, during the past few years has published results of great significance to forest pathologists and of considerable interest to all plant pathologists. In "Die Blaufäule des Nadelholzes" **5**: 531–573, 1907; **6**: 32–47, 297–323, 1908, he finds that the bluing of coniferous timber which has been quite generally ascribed to the action of *Ceratostomella pilifera* (Fries) is really caused by a series of *Ceratostomella* and closely related species, most of which he names and describes as new. Certain of these he finds to possess a Graphium stage of growth besides the conidial stage which Hedgcock (Studies Upon Some Chromogenic Fungi Which Discolor Wood, Report Mo. Bot. Garden **17**: 59–144. pls. 3–12, 1906 and in U. S. Dept. of Agr., Bureau of Plant Industry Bull. 36: 23–24, 1903) discovered and first described. Hedgcock also found that certain species of *Graphium* caused staining of wood, but in all his extensive culture work, covering nearly five years, he never found a *Ceratostomella* which possessed a Graphium stage. The discrepancy between the two workers should be thoroughly tested by a third investigator working with the cultures of both. Returning to Münch, we find that he concludes that *Ceratostomella* is parasitic. It readily attacks newly felled trees, but because of the high water content, cannot penetrate far. A loss of 10–20 per cent of water from winter-felled wood stimulates the growth so that it readily penetrates throughout the sapwood. In moist wood it grows mostly in the older layers of sapwood next the heartwood. If a living standing tree is free enough from water it can penetrate the sapwood also. Dr. von Tubeuf first called attention to the relation of air and water content to fungous attack in wood by *Merulius lacrymans*. In a second paper, "Untersuchungen über Immunität und Krankheitsempfänglichkeit der Holzpflanzen," **7**: 54–75, 87–114, 129–160, 1909, Münch extends this discovery to a number of different forms of fungi, with all of which it proves to hold true to a greater or less degree. *Armillaria mellea* proved to be able to thrive with relatively little air. Experiments were conducted with *Stereum purpureum* (Pers.), *Stereum hirsutum* (Willd.), *Collybia velutipes* Curt., *Hypholoma fasciculare* (Huds.), *Pholiota squarrosa* (Mull.), *P. adiposus Fr.*, *Schizophyllum commune* Fries., *Lenzites betulina* (L.), *Fomes igniarius* (L.), *F. fulvus* Scopoli, *F. fomentarius* L., *F. pinicola* (Swartz), *F. annosus* Fr., *Merulius lacrymans* (Wulf.), *Coniophora cerebella* (Alb. & Schw.),

Nectria cinnabarina (Tode) Fr., *N. ditissima* Tul., *Dasyscypha wilkommii* (Hartig), *Valsa sordida* Nitschke, and *V. leucostoma* (Pers). In this paper he also gives his methods of inoculating living trees with pure cultures of wood-rotting fungi. This is as follows: Chip off the rough bark of the tree at the desired place, wash this area with corrosive sublimate solution, dip bit into same solution and wash with absolute alcohol, bore hole, introduce pure culture material with instruments dipped in alcohol, close hole with a cork previously sterilized by dry heat in a flask and handled with pincers dipped in alcohol; wash cork and area around it with corrosive sublimate and cover with "Raupenleim". One small but important detail is not mentioned by Münch: The writer has found in his inoculations of wood blocks and trunks of trees that if the infection material (if not grown on agar, wet bread, or similar soft media) is wedged tightly against the wood to be infected, success is much more certain than when it is allowed to lie loosely against the block. In fact, this detail alone seemed very largely to determine the success of the inoculations. This has proved true of *Lenzites sepiaria* both in tube cultures and in field inoculations into green tree trunks, and with *Polystictus hirsutus* Fr., *P. pergamenus* Fr., *P. cinnabarina* Jacq., *P. versicolar* (L.) Fr., and *Lenzites betulina* Fr. in tube cultures. In a third paper, "Versuche über Baumkrankheiten" **8**: 389–408, 425–417, 1910, Münch gives the preliminary results of artificial inoculations of pure cultures of various fungi into living, healthy, tree trunks. Examinations at times varying from 3 months to 3¾ years showed the following results:

1. *Collybia velutipes* in horsechestnut formed sporophores.
2. *Schizophyllum commune* in *Fagus silvatica* formed discolored areas in the wood locally, but no sporophores.
3. *Stereum purpureum* in *Fagus silvatica* same as 2.
4. *Stereum rugosum* in *Fagus silvatica* discolored local areas.
5. *Stereum hirsutum* in *Fagus silvatica* same as 4.
6. *Fomes igniarius* in *Fagus silvatica* rotted the wood locally.
7. *Fomes fomentarius* in *Fagus silvatica* rotted the wood locally.

Apparently further papers are to be expected giving more complete results of these tests.

PERLEY SPAULDING

MINNEAPOLIS MEETING OF AMERICAN PHYTOPATHOLOGICAL SOCIETY (concluded)

A Study of Some Anthracnoses and their Relation to a Sweet Pea Disease. J. J. TAUBENHAUS

A serious disease was found to attack the sweet pea in Newark, Delaware. The injury was most apparent from the time of flowering to the maturing of the seeds. The disease was found to be due to a *Gloeosporium* sp. A pure culture of the organism was readily obtained, and many sweet pea seedlings successfully inoculated with it. Similar results were obtained by Sheldon, who inoculated sweet pea seedlings with a pure culture of *Glomerella rufo-maculans* (Berk.) Sp. & v. Sch., freshly obtained from a decaying apple. Inoculation with the original sweet pea *Gloeosporium* on the apple gave the typical bitter rot. The organism from the above affected apples was re-isolated and sweet pea seedlings were again successfully inoculated with it. This proved conclusively that the *Gloeosporium* of the sweet pea is the same as the *Gloeosporium fructigenum* Berk. of the apple.

It was also found that a species of *Gloeosporium* from the fruit of *Podophyllum peltatum*, the *Gloeosporium gallarum* Ch. Rich. and the *Gloeosporium officinale* Ell. & Ev., could also infect sweet pea seedlings and produce the same symptoms as that caused by the apple *Gloeosporium*, and the original sweet pea *Gloeosporium*. Futher investigations proved that the above three organisms could also produce the typical bitter rot on apples and pears. These evidences, together with morphological studies seem to indicate that the *Gloeosporium* from the *Podophyllum peltatum* fruit, the *Gleosporium officinale* Ell. & Ev. the *Gloeosporium gallarum* Ch. Rich. are one and the same with the *Glomerella rufo-maculans* (Berk.) Sp.& v.Sch. of the apple. It was also found that the above *Gloeosporiums* could readily produce a disease on pole lima beans and bush lima bean pods in the field.

The *Gloeosporium guava*, and the *Gloeosporium persea* failed over and over again to infect sweet pea seedlings, and to produce the typical bitter rot symptoms on the apple and pear.

The Ascogenous Form of the Fungus Causing Dead-Arm of the Grape. C. L. SHEAR

The ascogenous form of *Fusicoccum viticolum* Reddick is described. It is regarded as an undescribed species of *Cryptosporella* which is named *C. viticola*. It has been found associated with the pycnidial form on dead grapevines which had been attacked by the dead-arm disease. Pure cultures made from single ascospores produced pycnidial fructifications which correspond in every particular with *Fusicoccum viticolum* grown in pure cultures from pycnospores. Specimens of the ascogenous stage have been collected in New Jersey, Virginia and Michigan.

Further Notes on the Bark Diseases of the Chestnut. HAVEN METCALF and J. FRANKLIN COLLINS

Necrosis and Crown Gall Diseases of Grapes in New York. D. REDDICK.

The paper gives preliminary results of infection experiments to prove the pathogenesis of *Fusicoccum viticolum* Reddick. Inoculations of 18 months in various parts of the vine have failed to produce galls and it is thought that galls are caused by other agencies, among them, the crown gall organism, (*B. tumefaciens*). Lesions of *Fusicoccum* on shoots were abundant in 1910 owing to very favorable infection

weather early in the spring. Preserving such infected canes for bearing wood seems to be the chief means of entrance of the fungus into the canes and trunk. Necrosis appears to be much more abundant and destructive than Crown Gall.

Crown Gall is rarely responsible for the death of a vine. It usually occurs on stems and arms and has never been seen on roots. The variety Niagara is most affected.

Peach Leaf Curl in Nursery Stock. V. B. STEWART

Peach leaf curl caused by *Exoascus deformans*, in nursery stock is a very important disease from an economic standpoint and often causes heavy losses. The disease attacks the newly budded stock and the greater per cent of infected trees are injured to such an extent that they are unsalable.

There is a wide variation in the susceptibility of different varieties of peach stock to the leaf curl disease. In parallel nursery rows the following variation in susceptibility was noted., Elberta 23% ; Carman 22% ; Mt. Rose 13 % ; Globe 8.5 % ; Crosby 3.5 % ; Salway 2% ; Alexander 1%.

In order to control leaf curl in the nursery, each bud requires individual attention. This is necessary owing to the fact that the bud which is inserted in the seedling stock is more or less protected. The buds are inserted near the ground on the trunks of the seedlings. Their position often makes it difficult to thoroughly cover them with the spray mixture.

Some Laboratory and Field Studies on Fungicidal Values. E. WALLACE, F. M. BLODGETT, and LEX R. HESLER

This paper briefly summarizes the results of a practical application of a method of determining the fungicidal value of a spray mixture largely as described by Dr. Donald Reddick and the senior author of this paper in an article read at the first annual meeting of this society in December, 1909. This method was applied to a study of the various modifications of lime-sulfur solution and mixtures. Special attention was given to the effect of various modifications such as the addition of lead arsenate, precipitation by carbon dioxid, addition of lime and of iron sulfate, the value of sediment and its various constitutents, such as calcium sulfate, magnesium oxid and free sulfur. The results in several cases were checked up by actual spraying experiments which to a remarkable degree, confirmed the conclusions drawn from laboratory tests. A brief study of the principle of fungicidal action was also made by a special adaptation of this method, which seemed to indicate that the spore itself, is, in some way, probably by means of solvent excretions, active in bringing into solution small quantities of the fungicide which in turn prevents germination.

Varietal Resistance of Beans to Anthracnose. M. F. BARRUS

During the summer of 1910 one hundred and sixty-one varieties of beans were tested for their resistance to Anthracnose. There were seven different inoculations made throughout the summer on many of the varieties; four being on full grown plants in the field and under field conditions; two others on seedling plants, out of doors and shaded after inoculation ; and in the remaining case on seedlings of those varieties which had thus far proved resistant, or nearly so. The seedlings were grown in pots in the greenhouse and covered with lamp chimneys, plugged with cotton at the upper end. The inoculations were made with a suspension of spores, sprayed on plants with an ordinary hand sprayer. The plants in the field were inoculated at a time when the weather was rainy and continued misty for a day or so after the inoculation was made. The results show that, to the strain of fungus used, a few wax varieties were entirely resistant as well as most

of the Limas, and a few varieties were very nearly so. The larger number of varieties were quite susceptible and fair infection was obtained on a few varieties of Lima Beans on stems, pods and leaves.

A Preliminary Report upon the Effects of Arsenical Compounds upon Apple Trees. B. D. SWINGLE Published in full in PHYTOPATHOLOGY, No. 3

Results of Cotton Anthracnose Work for 1910. H. W. BARRE

Notes on Some Diseases of Trees in our National Forests, II. GEORGE GRANT HEDGCOCK. Read by title

A *New Fruit Spot of Apple.* W. M. SCOTT. Published in PHYTOPATHOLOGY No. 1.

A Contribution to our Knowledge of Mine Fungi. C. J. HUMPHREY

The paper gives a general survey of the conditions which favor the destruction of mine timber, with particular reference to the fungi. Conditions within the mines are fairly uniform throughout the year. The worst decay occurs where the air is moderately warm and humid. Where very humid the mycelium tends to grow superficially. Saturated timber contains too little oxygen to support growth while some of the driest places furnish insufficient moisture.

The preponderance of the vegetative stage is very marked. Infection within the mines occurs by the spread of the mycelium, which is often accompanied by rhizomorphs, and by the dispersal of spores from the mature fruiting bodies.

Fomes annosus Fr. is probably the most destructive coniferous fungus encountered. *Paxillus pannoides* Fr., *Polystictus versicolor* Fr., and this species constitute the three having the widest distribution in the United States. *Stereum fasciatum* Schw., *Lenzites betulina* Fr., *Coniophora cerebella* Fr., and *Merulius lachrymans* Fr. are also reported. The latter is very destructive to Engelmann spruce at Cripple Creek, Colo.

A Bacterial Disease of Bananas and Plantains. JAMES BIRCH RORER Published in full in PHYTOPATHOLOGY, No. 2

The Leaf Spot of Sugar Beets, C. O. TOWNSEND. Read by Title.

Notes on Hypertrophied Structures. MEL T. COOK

A comparison of the structure of the cecidia caused by insects and parasitic fungi, especially the Uredineae. The primary structural characters are practically the same. The progress of the development is very similar but the insect cecidia reach a much higher degree of complexity than the myco-cecidia. The highest myco-cecidia were similar to the diptro-cecidia, caused by insects of the genus Cecidomyia.

The Grand Rapids Tomato Disease. ERWIN F. SMITH

Grown Gall of Plants. ERWIN F. SMITH. Published in PHYTOPATHOLOGY, No. 1

Cereal Smut Spore Germination. E. C. STAKMAN. Read by title

Alfalfa Rust and Timothy Rust in Iowa. L. H. PAMMEL

C. L. SHEAR,
Secretary-Treasurer.